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New onset alcohol use disorder following bariatric surgery

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Abstract

Background Bariatric surgery is the most effective treatment for morbid obesity; however, there may be significant unanticipated psychosocial effects following surgery. Prior studies have identified a threefold increase in the incidence of alcohol use disorder (AUD) after Roux-en-Y gastric bypass (RYGB). With sleeve gastrectomy (SG) now comprising over 50% of primary bariatric operations, the degree to which patients who undergo SG develop AUD remains unknown. We sought to characterize the patients and incidence of AUD following SG compared to RYGB.

Methods This study used prospectively collected data from a state-wide quality collaborative. The presence of AUD was determined using the Alcohol Use Disorders Identification Test for Consumption (AUDIT-C), with a score ≥ 4 in men and ≥ 3 in women suggestive of AUD. We used bivariate Chi-square tests for categorical variables and independent samples *t* tests for continuous variables. We used multivariable logistic regression to identify patient characteristics that may predispose patients to development of AUD at 1 and 2 years after surgery.

Results The overall prevalence of AUD in our population ($n = 5724$) was 9.6% preoperatively, 8.5% at 1 year postoperatively, and 14.0% at 2 years postoperatively. The preoperative, 1-year, and 2-year prevalence of AUD for SG were 10.1%, 9.0%, and 14.4%, respectively. The preoperative, 1-year, and 2-year postoperative prevalence of AUD for RYGB were 7.6%, 6.3%, and 11.9%, respectively. Predisposing patient factors to AUD development included higher educational level ($p < 0.01$) and higher household income ($p < 0.01$).

Conclusions This is first large, multi-institutional study of AUD following SG. The prevalence of alcohol use disorder in patients undergoing SG and RYGB was similar pre- and postoperatively. The majority of patients developed AUD following their second postoperative year. Understanding the timing and incidence of alcohol use disorder in patients undergoing SG—the most commonly performed bariatric operation in the United States—is critical to providing appropriate counseling and treatment.

Keywords Bariatric surgery · Alcohol use disorder · Substance use

Bariatric surgery has proven to be a powerful tool to combat the global obesity epidemic [1]. Each year more patients pursue bariatric surgery as an effective means to lasting weight reduction [2]. While these patients often benefit from improvements in cardiovascular health, weight-related comorbidities, and quality of life measures, they are also subject to numerous unintended consequences such as X, Y, and Z [3, 4]. What is the hypothesized causal pathway here (in one or two sentences)? With the ever-increasing popularity of bariatric surgery, there is even more of an impetus to understand the postoperative complications that can arise, including the development of new substance use habits. A recent, multicenter study has indicated that the 5-year incidence of AUD in patients who undergo Roux-en-Y gastric bypass (RYGB) and laparoscopic adjustable gastric banding (LAGB) is 20.8% and 11.3%, respectively [5]. Similarly, other studies in this area have been limited in focus RYGB and LAGB [6–8]. In recent years, national trends in bariatric surgery demonstrate that LAGB has fallen out of favor and the sleeve gastrectomy (SG) procedure is on the rise

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[2]. SG currently accounts for the majority of bariatric surgeries done in the US today, yet there is very little in the literature on the development of substance use habits after this operation.

As such, the degree to which AUD occurs after SG remains unknown. The association between these bariatric surgeries and AUD is thought to be multifactorial in origin, both physiological and psychological. For example, in post-RYGB patients, the new anatomy has been shown to alter the metabolism of alcohol such that both peak blood alcohol content and return time to sobriety are increased [9, 10]. Moreover, studies in animal models have demonstrated that post-surgical hormonal changes may result in a reinforcement of the rewarding effects of alcohol [11]. Other studies have revealed associations between the neural pathways that fuel drug addiction and compulsive eating [12]. Based on neurobiological and clinical findings, some theorize that bariatric surgery patients may be susceptible to 'addiction transfer' or 'cross addiction,' whereby patients substitute alcohol use for binge eating after surgery [6]. Ultimately, AUD can contribute to significant long-term health consequences in bariatric surgery patients, including increased risk of critical illness, increased rates of hospitalization, and worse outcomes [13, 14]. It also presents an enormous burden of disease to health care systems worldwide [15]. As such, it is important that we characterize the risk of this outcome occurring in patients who have undergone SG, where some posit that the lack of gastrointestinal bypass should not affect alcohol consumption.

This study seeks to address the gaps in the literature by looking at the relative development of AUD postoperatively in SG and RYGB. We utilized a clinical registry of prospective, patient-reported and chart abstracted data from 40 hospitals in the state of Michigan. We evaluated 1 and 2 years rates of AUD in patients who have undergone SG or RYGB and differences in patient demographics associated with each group.

Methods

Study sample

This study used data from the Michigan Bariatric Surgery Collaborative (MBSC) registry, which is a voluntary consortium of hospitals and surgeons designed to gather data to improve the quality of care of patients undergoing bariatric (weight loss) surgery, as described and utilized in previous work [16]. Nearly 95% of patients having bariatric surgery in the state of Michigan are included in the clinical outcomes registry from these 40 hospitals. Trained data abstractors and healthcare providers are annually reassessed for accuracy and consistency by MBSC program staff. Use of the secondary, deidentified data were deemed exempt by the institutional review board of the University of Michigan.

Prior to inclusion in this registry, patients agree to complete a survey before surgery and annually for the next 5 years postoperatively. The surveys are either sent by mail or electronically with frequent reminders, including phone calls, in order to minimize the number of patients lost to follow up. These surveys assessed patient's alcohol use using the Alcohol Use Disorders Identification Test for Consumption (AUDIT-C) measure [18], as well as demographic information (sex, age, race, education, employment status and income), body mass index (BMI), and comorbidities. We identified all patients in the MBSC registry that underwent Roux-en-Y gastric bypass (RYBG), sleeve gastrectomy, laparoscopic banding, and duodenal switch (BPD-DS) from 2014 to 2017. Although data were also collected for laparoscopic banding ($n=25$) and duodenal switch surgeries ($n=29$), they were not included in our analysis due to limited sample size.

Outcomes

Our primary outcome for this study was the prevalence of harmful or hazardous drinking, indicative of Alcohol Use Disorder (AUD) before and after bariatric surgery. The AUDIT-C was used to identify that patients are drinking at levels that indicate they may have active alcohol use disorders. This is a three-question screening test to identify risky alcohol use behaviors in the prior 12 months. The AUDIT-C screening test has been widely tested and validated [17, 18].

Baseline AUDIT-C scores were assessed using a preoperative MBSC survey for each of the operations. The same measures were then reassessed every 12 months postoperatively through a written survey in the mail. Each of the responses to the three-question screening test is assigned a value ranging from 0 to 4 and totals were calculated for each patient (Fig. 1). An AUDIT-C score (range 0–12) of 4 points or more in men or 3 points or more in women suggests harmful and hazardous alcohol use, and possible dependence [18]. Accordingly, we assigned men with an AUDIT-C score ≥ 4 and women with an AUDIT-C score ≥ 3 as having AUD symptoms.

Other independent variables including sociodemographic information, smoking history, comorbidities and depression were self-reported and gathered preoperatively. Descriptions of these variables have been previously described [16].

Statistical analyses

In order to determine the statistical significance of differences in patient characteristics between procedure types and larger trends in the data across time periods and including all patients with AUDIT-C data we employed bivariate

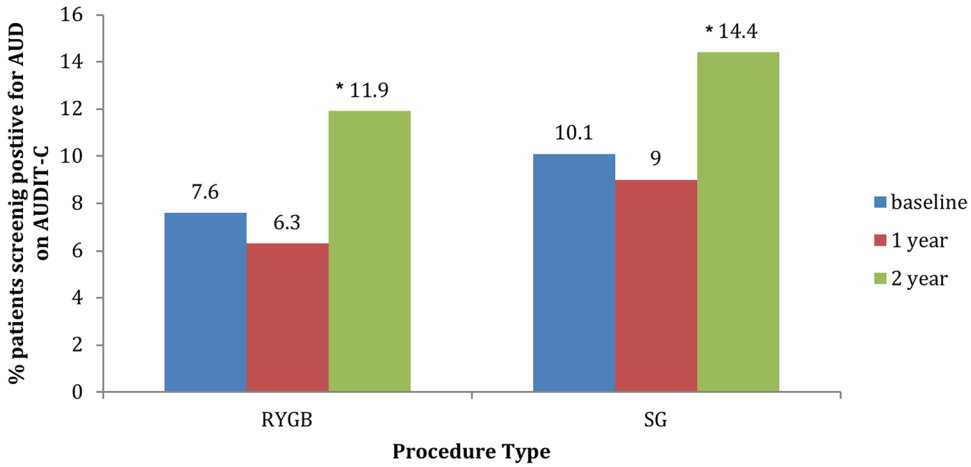

Fig. 1 Comparison of patients screening positive for AUD at baseline, year 1, and year 2. There is a significant increase in AUD positive patients at year 2 compared to baseline for RYGB and SG procedures (*$p < 0.001$)

Chi-square tests for categorical variables and independent samples *t* tests for continuous measurement variables. We used multivariable logistic regression to determine whether patient characteristics differed between patients that were positive for AUD at baseline and patients that were negative for AUD at baseline. This regression was also used to establish the relationship between patients without baseline AUD and AUD status at year 1 and year 2 and to produce adjusted odds ratios for various factors included in these models. All reported *p* values are 2-sided, and $p < 0.05$ was considered statistically significant. All statistical analyses were conducted with SAS 9.4 64-bit.

Results

This study surveyed 5724 patients who underwent either SG ($n = 4718$) or RYGB ($n = 1006$) and completed both baseline and 1 year postoperative evaluations. Of these patients, 1381 (24.1%) completed 2 years postoperative evaluations. We did not find any significant differences in the patient populations for whom follow-up data were available and those that were not. Comparisons of the baseline sociodemographic characteristics between the two procedures are presented in Table 1.

The percentage of patients with AUD symptoms following RYGB increased from 7.6% at baseline to 11.9% at 2 years postoperatively ($p = 0.02$), while the percentage of patients with AUD symptoms following SG increased from 10.1% at baseline to 14.4% at 2 years postoperatively ($p < 0.0001$). Both procedures revealed a decrease in the percentage of patients with AUD symptoms between baseline and 1 year postoperatively. These results are seen in Fig. 1.

The percentage of patients with new onset AUD during the first postoperative year was 0.54% and 0.75% for RYGB and SG, respectively. The percentage of new onset AUD for during the second postoperative year was 7.2% and 8.5% for RYGB and SG, respectively. These results are highlighted in Fig. 2.

The multivariable regression revealed patient characteristics associated with significantly increased odds of having AUD at 2 years post-RYGB to be income of $45,000–$74,999 (OR 14.02) or >$75,000 (OR 43.14). Characteristics associated with significantly decreased odds were lack of baseline AUD (OR < 0.001), no baseline alcohol consumption (OR 0.07), completing some college (OR 0.04), and having a college degree (OR 0.04). These results, along with characteristics not associated with changes in AUD prevalence, are presented in Table 2a.

No patient characteristics in the multivariable regression were associated with significantly increased odds of having AUD at 2-year post-SG. However, characteristics with significantly decreased odds of having AUD at 2-year post-SG were absence of baseline AUD (OR 0.001), no baseline alcohol consumption (OR 0.06), and the presence of comorbid depression (OR 0.50). These results are seen in Table 2b.

Discussion

This is the first study to prospectively assess the incidence of alcohol use disorder (AUD) in patients following SG. We found a similar prevalence of AUD at 1 and 2 years postoperatively in patients who underwent SG (1 year = 9%, 2 years = 14%) and RYGB (1 year = 6.3%, 2 years = 11.9%). We observed a small, not statistically significant increase in the development of new onset AUD in the first postoperative year for both SG (0.75%) and RYGB (0.54%). In the second postoperative year, there were statistically significant increases in the development of new onset AUD for both SG (8.5%) and RYGB (7.2%). Several predisposing patient factors to AUD development for patients undergoing RYGB included higher household income, lower educational level, and any baseline alcohol consumption. For SG patients, any

Table 1 Baseline characteristics RYGB to SG patients who completed AUDIT-C at baseline and year 1

Sociodemographic characteristics	Total	RYGB $N = 1006$	Sleeve $N = 4718$	p-value
Female, no. (%)	4489 (78.4)	791 (78.6)	3698 (78.4)	0.8624
Male, no. (%)	1235 (21.6)	215 (21.4)	1020 (21.6)	
Age (years), median (IQR)	46.2 (37.9–55.6)	46.9 (38.6–56.2)	46.0 (37.8–55.4)	0.0496
Race, no. (%)				
White	4707 (83.8)	847 (85.6)	3860 (83.4)	0.0483
Black	813 (14.5)	125 (12.6)	688 (14.9)	
Asian	13 (0.2)	5 (0.5)	8 (0.2)	
Other	85 (1.5)	12 (1.2)	73 (1.6)	
Hispanic ethnicity no. (%)	245 (4.3)	38 (3.8)	207 (4.4)	0.3965
Education, no. (%)				
≤ High school	1115 (19.6)	239 (23.9)	876 (18.7)	0.0008
Some college	1662 (46.9)	448 (44.8)	2214 (47.3)	
≥ College degree	1902 (33.5)	312 (31.2)	1590 (34.0)	
Employment status no. (%)				
Employed	3847 (67.9)	614 (61.7)	3233 (69.2)	<0.0001
Unemployed	213 (3.8)	47 (4.7)	166 (3.6)	
Disabled	616 (10.9)	143 (14.4)	473 (10.1)	
Other	988 (17.4)	191 (19.2)	797 (17.1)	
Household income				
< $25,000	1212 (21.9)	255 (26.5)	957 (20.9)	0.0002
< $25,000–$44,999	1170 (21.1)	211 (21.9)	959 (21.0)	
< $45,000–$74,999	1498 (27.0)	250 (26.0)	1248 (27.3)	
≥ $75,000	1659 (30.0)	247 (25.7)	1412 (30.9)	
BMI, median (IOR)	46.0 (41.6–51.9)	47.2 (42.2–52.9)	45.8 (41.5–51.7)	0.0009
Alcohol consumption @ baseline				
None	3106 (54.3)	630 (62.6)	2476 (52.5)	<0.001
Any	2208 (38.6)	318 (31.6)	1890 (40.1)	
Regular (≥ 2 times/week)	410 (7.2)	58 (5.8)	352 (7.5)	
Tobacco use				
No, never smoked	4217 (74.5)	728 (72.8)	3489 (74.9)	0.2178
Former smoked	1205 (21.3)	233 (23.3)	972 (20.9)	
Yes/current	238 (4.2)	39 (3.9)	199 (4.3)	
Comorbidities				
Depression	2556 (44.7)	466 (46.3)	2090 (44.3)	0.2411
Diabetes	1803 (31.5)	411 (40.9)	1392 (29.5)	<0.0001
Hypertension	2951 (51.6)	568 (56.5)	2383 (50.5)	0.0006
CAD	293 (5.1)	65 (6.5)	228 (4.8)	0.0333
CVD	3053 (53.3)	589 (58.6)	2464 (52.2)	0.0003
Serious lung disease	1475 (25.8)	318 (31.6)	1157 (24.5)	<0.0001
PUD	197 (3.4)	51 (5.1)	146 (3.1)	0.0018
3 or more comorbidities	2471 (43.2)	517 (51.4)	1954 (41.4)	<0.0001

baseline alcohol consumption was associated with higher AUD risk, while a preoperative diagnosis of depression was associated with lower AUD risk.

Existing literature has established undergoing RYGB as a risk factor for development of AUD following bariatric surgery [1, 6, 19, 20]. However, little data exist on the development of AUD in patients following SG [21]. A recent, multicenter study found that 20.8% of patients undergoing RYGB and 11.3% of patients undergoing LAGB develop AUD in the 5 years following surgery [5]. Additional studies found that RYGB is associated with accelerated alcohol absorption, higher peak blood alcohol concentration, and longer time to eliminate alcohol compared to before surgery [9, 10, 22]. The data are less clear on the pharmacokinetics

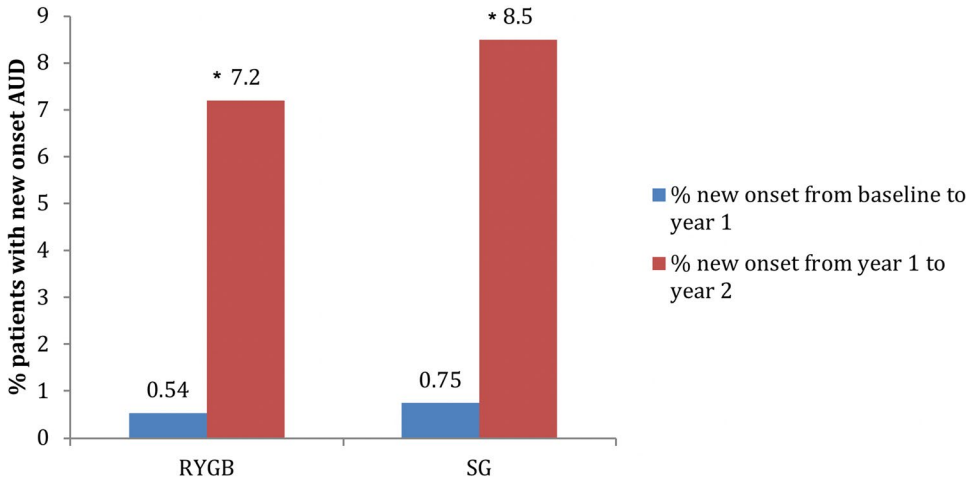

Fig. 2 Comparison of patients screening positive for AUD between baseline and year 1 and year 1 to year 2 following surgery

of alcohol absorption in patients following SG [23, 24]. Based on the fundamental difference in how RYGB and SG are formed, there is no alteration in gastrointestinal anatomy that may contribute to differences in absorption patterns, we hypothesized that patients undergoing SG would be at decreased risk for AUD development. However, our findings did not support this hypothesis and but are consistent with recent work suggesting that alterations in alcohol pharmacokinetics are unlikely to be the only explanation for increased alcohol consumption, with changes in brain reward processing also playing a role [25]. Our novel data demonstrated that SG patients face similar risks of developing AUD as RYGB patients. Next, it is important to understand when AUD develops after surgery.

A previous prospective cohort study reported an increase in the prevalence of AUD in patients after all bariatric surgical procedures that did not reach statistical significance until the second postoperative year [5]. Our study reinforced this finding, showing a marked increase in AUD incidence during the second postoperative year. By confirming this temporal pattern in the development of AUD for SG as well as RYGB, we may inform timely screening of these patients. The similarity in timing and incidence of new onset AUD in patients undergoing SG and RYGB also lends itself to further study on the mechanism of this phenomenon. As discussed in patients who have undergone other bariatric procedures, patients who undergo SG may be subject to a combination of physiological and psychological factors that predispose to AUD in the years following surgery [26]. It is thus important to preoperatively identify patients who may be at increased risk for development of AUD.

Identifying patient characteristics that predispose to increased AUD risk following surgery may allow for targeted screenings and counseling. We found that for RYGB patients, higher household income, lower educational level, any baseline alcohol consumption, and baseline AUD were associated with increased risk of AUD 2 years postoperatively. For SG patients, any baseline alcohol consumption and baseline AUD were associated with increased risk of AUD 2 years postoperatively. Interestingly in SG patients, we observed a decreased risk of AUD in patients with baseline depression. Perhaps patients with depression were more likely to have regular contact with mental health providers that screen for early signs of substance abuse, thus protecting against progression to AUD. These findings add to previously identified risk factors from the Longitudinal Assessment of Bariatric Surgery (LABS-2). Their prospective study identified preoperative alcohol consumption, smoking, less social support, younger age, and male gender as characteristics associated with increased AUD risk [5]. Similarly, the Swedish Obese Subjects study identified male gender, baseline smoking, and baseline alcohol consumption as risk factors for increased alcohol consumption [6]. Both of these large studies failed to include SG patients in their analysis. Thus, our data build on their work by including risk factors for the development of AUD specifically in SG patients.

Several limitations of our study warrant consideration. First, our data were obtained via the voluntary completion of an annual survey, so patients lost to follow up have the potential to skew our results. However, with prospective data obtained using a thorough and frequent follow-up strategy, we were able to capture a high percentage of patients (45%) at 2 years postoperatively. With respect to a large clinical registry such as the MBSC, previously published work cites rates above 40% as excellent [27]. Second, our data were obtained from surveys in which patients self-report alcohol consumption, which may result in some inaccuracies regarding true alcohol consumption. However, inaccuracies in reporting would bias our results towards the null, as patients are likely to underreport true alcohol consumption [28]. Third, our data are from a state-wide quality collaborative that represents patients and outcomes only within the state of Michigan, which may not be representative of

Table 2 Multivariable logistic regression analyses comparing patients that are AUD positive at baseline, year 1, and year 2 to patients that are AUD negative at baseline following Roux-en-Y gastric bypass (a) and Sleeve gastrectomy (b)

(a) Baseline characteristics of adults with and w/o AUD following RYGB

Sociodemographic characteristics	No AUD baseline $N=930$	AUD baseline $N=76$	Adjusted OR (95% CI)	1 year AUD $N=63$	OR (95% CI)	2 year AUD $N=81$	OR (95% CI)
Baseline AUD	–	–	–	58 (92.1)	Ref.	76 (93.8)	Ref.
Baseline no. AUD	–	–	–	5 (7.9)	< 0.001 (< 0.001, 0.006)	5 (6.2)	< 0.001 (< 0.001, 0.009)
Female no. (%)	731 (78.6)	60 (78.9)	4.64 (1.13, 19.02)	49 (77.8)	0.26 (0.06, 1.20)	16 (19.8)	
Male no. (%)	199 (21.4)	16 (21.1)	Ref.	14 (22.2)	Ref.	65 (80.2)	
Age (years) Median (IQR)	47.0 (39.0–56.4)	45.9 (35.3–55.0)	0.95 (0.90, 0.99)	46.2 (35.1–54.9)	0.99 (0.93, 1.06)	46.8 (38.4, 57.4)	
Race no. (%)							
White	780 (85.4)	67 (88.2)	Ref.	54 (85.7)	Ref.	74 (91.4)	
Non-White	133 (14.6)	9 (11.8)	0.53 (0.15, 1.80)	9 (14.3)	3.50 (0.55, 22.36)	7 (8.6)	
Hispanic ethnicity no. (%)	36 (3.9)	2 (2.6)	0.31 (0.03, 3.58)	2 (3.2)	1.84 (0.01, 374.85)	4 (5.0)	
Education no. (%)							
≤ High school	223 (24.2)	16 (21.1)	Ref.	16 (25.4)	Ref.	14 (17.3)	
Some college	419 (45.4)	29 (38.2)	0.69 (0.22, 2.22)	26 (41.3)	0.79 (0.14, 4.55)	34 (42.0)	
≥ College degree	281 (30.4)	31 (40.8)	0.54 (0.15, 1.95)	21 (33.3)	0.23 (0.04, 1.49)	33 (40.7)	
Employment status no. (%)							
Employed	561 (61.0)	53 (69.7)	Ref.	40 (63.5)	Ref.	64 (79.0)	Ref.
Unemployed	42 (4.6)	5 (6.6)	3.20 (0.51, 20.16)	4 (6.4)	1.52 (0.09, 27.00)	2 (2.5)	0.19 (0.01, 4.81)
Disabled	135 (14.7)	8 (10.5)	2.73 (0.52, 14.06)	10 (15.9)	16.60 (1.53, 180.45)	4 (4.9)	0.77 (0.01, 43.26)
Other	181 (19.7)	10 (13.2)	0.42 (0.09, 1.90)	9 (14.3)	4.47 (0.55, 36.45)	11 (13.6)	5.51 (0.73, 41.60)
Household income							
< $25,000	246 (27.7)	9 (12.0)	Ref.	10 (15.9)	Ref.	4 (5.1)	Ref.
$25,000–$44,999	197 (22.2)	14 (18.7)	2.33 (0.49, 11.06)	12 (19.1)	0.94 (0.10, 9.32)	16 (20.3)	5.83 (0.42, 81.32)
$45,000–$74,999	230 (25.9)	20 (26.7)	3.63 (0.77, 17.06)	17 (27.0)	1.85 (0.21, 16.68)	22 (27.8)	14.02 (1.03, 190.31)
≥ $75,000	215 (24.2)	32 (42.7)	2.11 (0.39, 11.52)	24 (38.1)	0.88 (0.09, 8.50)	37 (46.8)	43.14 (3.00, 620.17)
Baseline BMI, median (IQR)	47.3 (42.3–53.0)	44.9 (39.9–51.8)	0.92 (0.87, 0.98)	44.8 (38.8–51.7)	0.94 (0.86, 1.03)	45.2 (41.3, 50.0)	0.96 (0.87, 1.07)
Baseline alcohol consumption, no. (%)							
None	630 (67.7)	0 (0.0)	< 0.001 (< 0.001, > 999.99)	2 (3.2)	0.10 (0.01, 1.19)	1 (1.2)	0.07 (0.01, 0.91)
Any	290 (31.2)	28 (36.8)	Ref.	23 (36.5)	Ref.	20 (24.7)	Ref.
Regular (≥ 2 times/week)	10 (1.1)	48 (63.2)	259.53 (61.61, > 999.99)	38 (60.3)	2.19 (0.41, 11.59)	60 (74.1)	3.68 (0.50, 27.28)
Tobacco use							
No, never smoked	683 (73.8)	45 (60.8)	0.86 (0.17, 4.39)	40 (64.5)	3.50 (0.28, 44.52)	49 (62.0)	4.20 (0.48, 36.64)

Table 2 (continued)

(a) Baseline characteristics of adults with and w/o AUD following RYGB

Sociodemographic characteristics	No AUD baseline $N=930$	AUD baseline $N=76$	Adjusted OR (95% CI)	1 year AUD $N=63$	OR (95% CI)	2 year AUD $N=81$	OR (95% CI)
Former smoker	208 (22.5)	25 (33.8)	3.13 (0.55, 17.69)	19 (30.7)	1.07 (0.07, 15.71)	24 (30.4)	9.89 (0.91, 107.08)
Yes/current	35 (3.8)	4 (5.4)	Ref.	3 (4.8)	Ref.	6 (7.6)	Ref.
Comorbidities							
Depression	427 (45.9)	39 (51.3)	0.55 (0.22, 1.40)	33 (52.4)	0.55 (0.14, 2.26)	40 (49.4)	1.70 (0.39, 7.42)
Diabetes	391 (42.0)	20 (26.3)	1.37 (0.50, 3.76)	13 (20.6)	4.44 (0.96, 20.57)	29 (35.8)	0.86 (0.17, 4.34)
Hypertension	525 (56.5)	43 (56.6)	0.15 (<0.001, 42.18)	31 (49.2)	95.88 (5.95, >999.99)	50 (61.7)	<0.001 (<0.001, >999.99)
CAD	60 (6.5)	5 (6.6)	1.85 (0.28, 12.23)	4 (6.4)	4.00 (0.31, 51.05)	5 (6.2)	0.38 (0.01, 10.66)
CVD	545 (58.6)	44 (57.9)	4.24 (0.02, >999.99)	34 (54.0)	0.01 (<0.001, 0.18)	50 (61.7)	>999.99 (<0.001, >999.99)
Serious lung disease	304 (32.7)	14 (18.4)	3.35 (1.20, 9.30)	12 (19.1)	1.63 (0.34, 7.78)	17 (21.0)	0.86 (0.16, 4.77)
PUD	49 (5.3)	2 (2.6)	0.91 (0.11, 7.96)	2 (3.2)	0.30 (0.01, 10.73)	4 (4.9)	0.39 (0.01, 32.63)
3 or more comorbidities	481 (51.7)	36 (47.4)	0.85 (0.16, 4.61)	26 (41.3)	1.76 (0.19, 16.53)	48 (59.3)	0.39 (0.02, 6.95)

(b) Baseline characteristics of adults with and w/o AUD following SG

Sociodemographic characteristics	Baseline $N=4242$	AUD baseline $N=476$	Adjusted OR (95% CI)	1 year AUD $N=423$	OR (95% CI)	2 year AUD $N=365$	OR (95% CI)
Baseline AUD	–	–	–	391 (92.4)	Ref.	345 (94.5)	Ref.
Baseline no. AUD	–	–	–	32 (7.6)	0.003 (0.002, 0.005)	20 (5.5)	0.001 (<0.001, 0.003)
Female no. (%)	3331 (78.5)	367 (77.1)	3.16 (1.93, 5.15)	325 (76.8)	0.86 (0.50, 1.48)	281 (77.0)	0.49 (0.23, 1.05)
Male no. (%)	911 (21.5)	109 (22.9)	Ref.	98 (23.2)	Ref.	84 (23.0)	Ref.
Age (years), median (IQR)	46.2 (38.1–55.7)	43.2 (35.2–53.2)	0.97 (0.95, 0.98)	43.2 (35.2–53.2)	1.00 (0.98, 1.02)	45.9 (38.1, 54.6)	1.01 (0.98, 1.05)
Race no. (%)							
White	3461 (83.2)	399 (85.3)	Ref.	359 (86.1)	Ref.	322 (90.5)	Ref.
Non-White	700 (16.8)	69 (14.7)	0.51 (0.33, 0.82)	58 (13.9)	0.86 (0.48, 1.55)	34 (9.6)	0.98 (0.37, 2.59)
Hispanic ethnicity no. (%)	185 (4.4)	22 (4.7)	0.83 (0.39, 1.79)	21 (5.0)	0.58 (0.20, 1.72)	11 (3.1)	2.57 (0.54, 12.17)
Education no. (%)							
≤ High school	819 (19.5)	57 (12.1)	Ref.	47 (11.2)	Ref.	43 (11.9)	Ref.
Some college	1992 (47.3)	222 (46.9)	0.76 (0.48, 1.21)	195 (46.3)	1.23 (0.66, 2.40)	164 (45.4)	1.02 (0.40, 2.59)
≥ College degree	1396 (33.2)	194 (41.0)	0.73 (0.44, 1.21)	179 (42.5)	1.58 (0.79, 3.18)	154 (42.7)	1.80 (0.67, 4.81)
Employment status no. (%)							
Employed	2853 (68.0)	380 (80.5)	Ref.	340 (81.2)	Ref.	276 (75.8)	Ref.
Unemployed	154 (3.7)	12 (2.5)	1.51 (0.62, 3.67)	12 (2.9)	1.67 (0.45, 6.16)	6 (1.7)	0.39 (0.05, 2.87)

Table 2 (continued)

(b) Baseline characteristics of adults with and w/o AUD following SG

Sociodemographic characteristics	Baseline $N=4242$	AUD baseline $N=476$	Adjusted OR (95% CI)	1 year AUD $N=423$	OR (95% CI)	2 year AUD $N=365$	OR (95% CI)
Disabled	455 (10.8)	18 (3.8)	0.73 (0.33, 1.65)	10 (2.4)	0.23 (0.08, 0.67)	24 (6.6)	1.43 (0.34, 5.95)
Other	735 (17.5)	62 (13.1)	0.81 (0.50, 1.31)	57 (13.6)	1.05 (0.56, 1.99)	58 (15.9)	0.88 (0.37, 2.09)
Household income							
< $25,000	894 (21.8)	63 (13.5)	Ref.	53 (12.8)	Ref.	48 (13.3)	Ref.
$25,000–$44,999	876 (21.3)	83 (17.8)	1.16 (0.68, 1.97)	77 (18.6)	1.08 (0.51, 2.31)	55 (15.3)	0.78 (0.25, 2.45)
$45,000–$74,999	1118 (27.2)	130 (27.8)	1.31 (0.78, 2.21)	109 (26.3)	0.67 (0.32, 1.38)	104 (28.9)	1.11 (0.37, 3.33)
≥ $75,000	1221 (29.7)	191 (40.9)	1.13 (0.66, 1.94)	175 (42.3)	0.95 (0.45, 2.03)	153 (42.5)	1.04 (0.33, 3.26)
Baseline BMI, median (IQR)	46.0 (41.7–51.9)	43.8 (40.5–50.4)	0.98 (0.96, 1.00)	43.8 (40.6–50.1)	0.99 (0.96, 1.02)	43.1 (39.7, 49.7)	0.97 (0.94, 1.01)
Baseline alcohol consumption no. (%)							
None	2475 (58.4)	1 (0.2)	0.01 (< 0.0001, 0.04)	4 (1.0)	0.11 (0.04, 0.33)	1 (0.3)	0.06 (0.01, 0.42)
Any	1721 (40.6)	169 (35.5)	Ref.	167 (39.5)	Ref.	131 (35.9)	Ref.
Regular (≥ 2 times/week)	46 (1.1)	306 (64.3)	149.14 (92.81, 239.65)	252 (59.6)	0.92 (0.54, 1.57)	233 (63.8)	0.80 (0.35, 1.82)
Tobacco use							
No, never smoked	3175 (75.9)	314 (66.2)	0.59 (0.33, 1.07)	274 (65.1)	0.54 (0.23, 1.29)	239 (66.4)	0.68 (0.18, 2.58)
Former smoker	852 (20.4)	120 (25.3)	0.73 (0.38, 1.39)	110 (26.1)	0.71 (0.28, 1.82)	100 (27.8)	0.53 (0.13, 2.14)
Yes/current	159 (3.8)	40 (8.4)	Ref.	37 (8.8)	Ref.	21 (5.8)	Ref.
Comorbidities							
Depression	1875 (44.2)	215 (45.2)	0.91 (0.65, 1.27)	188 (44.4)	1.04 (0.65, 1.67)	172 (47.1)	0.50 (0.25, 0.98)
Diabetes	1304 (30.7)	88 (18.5)	1.45 (0.94, 2.23)	80 (18.9)	1.06 (0.58, 1.93)	73 (20.0)	1.45 (0.66, 3.19)
Hypertension	2186 (51.5)	197 (41.4)	0.89 (0.27, 2.95)	175 (41.4)	0.66 (0.14, 3.22)	178 (48.8)	1.34 (0.19, 9.47)
CAD	211 (5.0)	17 (3.6)	1.33 (0.52, 3.37)	14 (3.3)	1.23 (0.38, 3.99)	16 (4.4)	1.65 (0.42, 6.44)
CVD	2259 (53.3)	205 (43.1)	1.38 (0.43, 4.47)	181 (42.8)	1.58 (0.33, 7.62)	186 (51.0)	0.59 (0.08, 4.13)
Serious lung disease	1068 (25.2)	89 (18.7)	1.13 (0.77, 1.67)	74 (17.5)	1.36 (0.80, 2.34)	73 (20.0)	0.50 (0.23, 1.07)
PUD	140 (3.3)	6 (1.3)	3.59 (1.08, 11.96)	5 (1.2)	1.71 (0.36, 8.15)	11 (3.0)	1.35 (0.27, 6.82)
3 or more comorbidities	1806 (42.6)	148 (31.1)	0.84 (0.45, 1.55)	130 (30.7)	1.00 (0.43, 2.29)	129 (35.3)	1.65 (0.52, 5.20)

bariatric surgery patients nationwide. However, the use of the MBSC containing 40 hospitals from different geographical and socioeconomic regions throughout the state provides a diverse range of practices and patients for analysis. It is unlikely that patients or practices in Michigan are fundamentally different from the rest of the country. Finally, we found 7–10% of patients screening positive for AUD at baseline preoperatively. A diagnosis of AUD would preclude most practices from performing bariatric surgery as we confirmed with members of our collaborative. It is unclear what

this represents, but it may be a population of patients who are underdiagnosed with AUD using our current conventions. The AUDIT-C represents a useful screening tool that could be incorporated into the otherwise structured preoperative assessment of patients considering bariatric surgery.

With the rise in popularity of SG as a leading option for bariatric surgery, it is important that patients and providers understand the risks of postoperative complications, including the development of AUD after surgery. Our findings demonstrated that 8.5% of patients undergoing SG develop new onset AUD in the 2 years after surgery, a serious complication that previously lacked data. With the multitude of psychosocial changes that accompany bariatric procedures, the 2 years following surgery may be a critical time for the development of new substance use behaviors [16, 29, 30]. Alcohol use should thus be a point of emphasis in preoperative planning for SG patients, as well as an integral part of postoperative follow-up appointments. Special screening attention in the second postoperative year may be especially useful, as this is the time frame during which we observed the marked increase in AUD symptoms. In addition, patients with any baseline alcohol consumption may be at increased AUD risk, thus potentially requiring special screening and follow-up attention. In RYGB, patients with higher household income and lower educational level also may be at increased AUD risk. Further work should explore the etiology of these risk factors in determining why these subgroups may be at higher risk for AUD and identify possible interventions.

Another notable finding of our study was that patients undergoing SG and RYGB were at a similar risk of AUD. This information could be referenced in preoperative discussions surrounding procedure choice to fully inform the patient of their risks. Furthermore, the similarity in AUD risk suggests that changes in reward processing at the level of the brain may be more responsible for changes in behavior than the altered gastrointestinal physiology that changes alcohol absorption. One theory supports addiction transfer may be at play, specifically with carbohydrates and alcohol is a concentrated source of carbohydrates. Directed research exploring the neuroendocrine changes that occur after bariatric surgery has the potential to explain these changes and perhaps guide pharmacologic intervention.

Conclusion

As treatment for severe obesity, patients undergoing SG are at a similar risk of developing AUD as patients undergoing RYGB at 2 years postoperatively. In RYGB patients, any baseline alcohol consumption, higher household income, and lower educational level were baseline characteristics associated with increased risk of AUD. In SG patients, any baseline alcohol consumption was associated with increased risk of AUD, while baseline depression was associated with lower AUD risk. Patients and providers should be aware of the AUD risks of each bariatric surgical procedure, and AUD screening efforts may be most effective during the second postoperative year. Understanding the role of altered brain reward processing in these patients will be critical to developing therapeutic interventions.

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Compliance with ethical standards

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